IV. Claims 7-8, drawn to a multiprotein complex, classified in class 530, subclass 350.

V. Claims 9-11, drawn to an antibody which binds a multiprotein complex, classified in class 530, subclass 387.1.

VI. Claims 12 and 15, drawn to an agent which binds to a multiprotein complex, unclassifiable as no physical characteristics have been defined to allow classification.

VII. Claim 13, drawn to an agent which inhibits or modulates the binding of human MTT1 to eRF3 or MTT1 to a polysome, unclassifiable as no physical characteristics have been defined to allow classification.

VIII. Claims 14 and 16, drawn to an antisense molecule which facilitates binding of human MTT1 to eRF3, or MTT1 to a polysome, classified in class 536, subclass 23.1.

IX. Claims 14 and 16, drawn to a ribozyme which facilitates binding of human MTT1 to eFR3 or MTT1 to a polysome, classified in class 435, subclass 91.31.

X. Claim 17, drawn to a method of modulating peptidyl transferase activity during translation with a multiprotein complex, classified in class 435, subclass 4.

XI. Claims 18-20, drawn to a method of modulating peptidyl transferase activity during translation with an agent which binds a multiprotein complex, classified in class 435, subclass 4.

XII. Claims 21-25, drawn to a method for screening for a drug using a multiprotein complex, classified in class 530, subclass 350.

XIII. Claims 26, drawn to a method of modulating the efficiency of translation termination of mRNA and/or degradation of aberrant transcripts with a multiprotein complex, classified in class 530, subclass 350.

XIV. Claim 27, drawn to a method for identifying a disease involving a defect in a multiprotein complex, classified in class 424, subclass 9.1.

XV. Claims 28-30, drawn to a method to treat a disease by administering a multiprotein complex or an agent which binds to a multiprotein complex, classified in class 514, subclass 2.

XVI. Claim 31, drawn to a method to identify a disease involving a defective multimeric protein, classified in class 424, subclass 9.1.

XVII. Claims 32-41, drawn to a method of identifying genes involved in modulation of translation termination using the motifs disclosed in SEQ ID NO:1-9; classified in class 436, subclass 9.

Applicants elect Group IV (claims 7 and 8), with traverse for reasons given below.

By way of review, one aspect of the invention relates to the discovery that Helicase B is involved in modulating translation termination. It is for this reason that the helicase was renamed as MTT1 by applicants (for Modulator of Translation Termination). Applicants have discovered that the MTT1 gene and its protein product (MTT1p) are involved in modulating translation termination at a nonsense codon, but are not required for nonsense mediated mRNA decay. In particular, applicants have found that cells lacking MTT1 demonstrate a nonsense suppression phenotype, and do not affect nonsense-mediated mRNA decay. Furthermore, Applicants have also discovered that MTT1p interacts with the peptidyl release factor eRF3, is polysome-associated and demonstrates RNA-dependent ATPase and helicase activities. These results, taken together with the homology of the MTT1 gene and its protein product with the UPF1, demonstrate that MTT1 is involved in modulating translation termination at nonsense codons. Such discoveries were not previously known, and have important implications for the treatment of diseases that arise as a consequence of nonsense mutations.

Applicants submit that at least Groups I-XVI are linked by the novel and inventive concept of MTT1 (the gene and its protein product) as a modulator of translation termination. Therefore, Applicants request that the Examiner reconsider the restriction of these groups and that claims 1-31 be examined together.

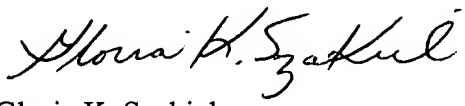
Moreover, by performing a complete search directed to any one of Groups I-XVI, the Examiner is likely to have also performed a search to Group XVII. With respect to the Group XVII claims, the MTT1 protein product includes all of the motifs to be a superfamily group I helicase (Figure 2), and the MTT1 gene (as well as its protein product) is involved in the modulation of translation termination. Therefore, a search directed to any one of Groups I-XVI

should substantially overlap with a search directed to Group XVII, and it would seem that consideration of the Group XVII claims would not place an undue burden of search on the Examiner.

For these reasons, Applicants respectfully request that the requirement for restriction by withdrawn and consideration of all of the claims on the merits be commenced.

Should the Examiner have any questions, the Examiner is respectfully invited to contact the undersigned agent at the telephone number set forth below.

Respectfully submitted,



Gloria K. Szakiel
Reg. No. 45,149
Agent for Applicant

HOFFMANN & BARON, LLP
6900 Jericho Turnpike
Syosset, NY 11791
(973) 331-1700